I. Amendments to the Specification:

Please amend the specification as shown:

Please delete the paragraphs on page 26, lines 13-35, and replace them with the following paragraphs:

construction of the plasmid pBBLnt: the *lgtA* gene present in the construct pCWlgtA (Gilbert *et al.*) was amplified by PCR at the same time as the UV5 tactac promoter of the plasmid using the primers CTTTAAGCTTCCGGCTCGTATAA **(SEQ ID NO: 1)** (sense, upstream promoter) and GACAGCTTATCATCGATAAGCTT **(SEQ ID NO: 2)** (antisense, lgtA end) both containing a *HindIII* site. The 1.3-kb amplified fragment was then subcloned into the *HindIII* site of the vector pBBlgtB.

construction of the plasmid pBBLntRcsA: The *rcsA* gene (Stout *et al.*, 1991) was first amplified by PCR starting with genomic DNA from JM 109 with the primers AGGGTACCCATGTTGTTCCGTTTAG **(SEQ ID NO: 3)** (*KpnI* site, *rcsA* left) and AATCTAGAGTAATCTTATTCAGCCTG **(SEQ ID NO: 4)** (*XbaI* site, *rcsA* right), and then cloned into the *KnpI-XbaI* sites of the vector pBBR1-MCS. The vector pBBR1-MCS-rcsA was then opened upstream of the gene by digestion with *KpnI*, rendered blunt (Amersham kit), freed with *XbaI* and inserted into the *SmaI-XbaI* sites of the construct pBBLnt, allowing a cloning downstream of the *lgtB-UV5tactac-lgtA* assembly, placing *rcsA* under the control of the UV5 tactac promoter.

Please delete the paragraphs on page 30, line 25, to page 31, line 28, and replace them with the following paragraphs:

A first 1.6-kb *BamHI-XbaI* fragment comprising the right-hand portion of *nanA* was amplified from the genomic DNA of JM 109 using the primers AAAGGATCCAAGATCAGGATGTTCACG **(SEQ ID NO: 5)** and GCTCTAGAATGGTAATGATGAGGCAC **(SEQ ID NO: 6)** and cloned between the *BamHI* and *XbaI* sites of the vector pUC19, forming the vector pUC-nan1.6. A second 2.1-kb *KpnI-BamHI* fragment comprising the left-hand portion of *nanA* was amplified using the

primers AAAGGATCCGCGTAGGTGCGCTGAAAC **(SEQ ID NO: 7)** and AAAGGTACCTCAGGCCACCGTTAGCAG **(SEQ ID NO: 8)** and cloned between the *KpnI* and *BamHI* sites of the vector pUC-nan1.6, forming the vector pUC-nan3.7. The kanamycin-resistance gene (pUC-4K, Pharmacia cassette) was then cloned into the *BamHI* site of pUC-nan3.7. The 4.9-kb *SacI-XbaI* fragment containing nanA::kan was inserted into the same sites of the suicide vector pCVD442 (Donnenberg and Kaper, 1991). This plasmid was used to obtain by homologous recombination JM 107 *nanA::kan* mutants, selected for their resistance to kanamycin and their inability to metabolize sialic acid (strain JM 107-nanA$^-$).

1.9. Construction of the strain JM 107col$^-$DE3

Suppression of the capacity to synthesize colanic acid was achieved by insertional inactivation of the *wcaJ* gene encoding a glucosyl-transferase (Stevenson *et al.*, 1996). A 1.8-kb DNA fragment containing the *wcaJ* gene and adjacent DNA were amplified by PCR starting with genomic DNA from JM 109, and inserted into a vector pTOPO2.1 (Invitrogen PCR cloning kit), with the aid of the primers CCACGATCCACGTCTCTCC **(SEQ ID NO: 9)** (right *wcaJ*) and AAGCTCATATCAATATGCCGCT **(SEQ ID NO: 10)** (left *wcaJ*). It was then transferred into a pUC19 vector into the *EcoRI* site. The vector thus obtained was subjected to a treatment with an *EcoR1* methylase, allowing the subsequent addition of the kanamycin-resistance gene to the ApoI site present at the center of *wcaJ*. The recombinant DNA *wcaJ::kan* was finally transferred into the suicide vector pCVD442 allowing, by homologous recombination, the production of JM 107 genomic mutants containing the inactivated gene, selected by PCR with the aid of the primers which were used for the cloning (strain JM 107-col$^-$).